

## Research Article

## Nasal Congestion and its Management in Pregnancy Rhinitis

*Hidung Tersumbat dan Penatalaksanaannya pada Rinitis Kehamilan*

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**Abstract**

**Objective :** To elaborate on the pathomechanism of pregnancy rhinitis and the proper management of rhinitis symptoms, particularly nasal obstruction.

Methods: Literature review.

**Methods :** Literature review.

**Result:** Placental Growth Hormone has a similar effect as progesterone in pregnancy, which is peripheral vasodilatation and increases extracellular volume. Increased estrogen during pregnancy enhances the parasympathetic activity, thus increasing vascular permeability and glandular activity. Plasma leakage from vascular bed to stroma results in edematous turbinates, causing nasal congestion. This mucosal swelling is exaggerated with the presence of thick and profuse secretion.

**Conclusions :** Pregnancy rhinitis, manifested as nasal congestion, is considered a phenomenon and may become a serious condition. Persistent nasal congestion acts as a potential risk factor in affecting fetal growth and development through gradual hypoxia process. This condition can lead to various complications such as maternal hypertension, preeclampsia, impaired fetal growth, and low APGAR scores. In-depth knowledge of pathomechanism is essential as guidance to proper treatment, including conservative and medical therapies, which will lead to an optimal outcome for both mother and baby.

**Keywords :** estrogen, nasal congestion, placental growth hormone, pregnancy rhinitis.

**Abstrak**

**Tujuan :** Untuk memaparkan patomekanisme rinitis kehamilan dan tatalaksana yang tepat dalam mengatasi gejala rinitisnya, terutama hidung tersumbat.

**Metode :** Tinjauan pustaka.

**Hasil:** PGH dan progesterone memiliki efek serupa yaitu vasodilatasi perifer dan peningkatan volume ekstraselular. Peningkatan estrogen selama kehamilan menstimulus aktivitas sistem parasimpatetik, yang mana terjadi peningkatan permeabilitas vaskular dan aktivitas kelenjar. Kebocoran plasma dari pembuluh darah ke stroma akan menyebabkan edema konka yang bermanifestasi sebagai kongesti hidung. Kondisi pembengkakan mukosa ini diperberat dengan adanya hipersekresi.

**Kesimpulan :** Rinitis kehamilan, dengan manifestasi kongesti hidung, dianggap sebagai suatu fenomena yang dapat menjadi fatal. Kongesti hidung persisten merupakan faktor risiko terjadinya gangguan tumbuh kembang janin melalui proses hipoksia bertahap. Kondisi ini dapat berlanjut menimbulkan komplikasi seperti hipertensi maternal, preeklamsia, gangguan tumbuh janin, dan skor APGAR yang rendah. Memahami patomekanisme sangat utama dalam membimbing klinisi memberikan tatalaksana yang tepat, termasuk terapi konservatif dan farmaka, yang akan memberikan keluaran yang optimal baik bagi ibu dan bayi.

**Kata kunci :** estrogen, kongesti hidung, placental growth hormone, rinitis kehamilan.

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**INTRODUCTION**

Rhinitis is a common problem during pregnancy, affecting up to 30% of pregnant women.<sup>1</sup> Rhinitis during pregnancy might occur as allergic rhinitis, drug-induced rhinitis, rhinosinusitis, anatomical variations, and gestational or "pregnancy" rhinitis.<sup>2</sup> In women with prior history of allergic rhinitis, nasal symptoms might improve or worsen during their pregnancies.<sup>3, 4</sup> Demoly<sup>1</sup> quoted that

around 10–30% pregnant women suffer more from their AR symptoms. However, some women experience rhinitis symptoms only a few episodes in their pregnancies. Thus, pregnancy rhinitis is defined as nasal congestion present during the last six weeks or more of pregnancy, without other signs of respiratory tract infection and no known allergic cause. Pregnancy rhinitis usually disappears within two weeks after delivery.<sup>1, 3</sup>

Pregnancy rhinitis is reported in one of five pregnant women.<sup>3</sup> The incidence rate from small groups of pregnant women were respectively 30% of 79 women, 18% of 66 women, and 21% of 160 women. The prevalence of pregnancy rhinitis as 17.17%, with the description as follows: 0% in the first trimester, 9.38% in the second trimester, and 38.89% in the third trimester.<sup>3,4</sup>

In a questionnaire study, 22% of 599 pregnant women reported having nasal congestion during their midwife visits. Rhinitis symptoms occurred in their 7<sup>th</sup> to 36<sup>th</sup> week of pregnancies and disappeared in second to the fourth week after delivery. Forty two percent of 2.264 pregnant women had nasal obstruction on the 36<sup>th</sup> week of pregnancies.<sup>3</sup>

Pregnancy rhinitis is considered as the result of hormonal changes and fluctuation during pregnancy, which might manifest as nasal congestion as the most bothersome symptom. During pregnancy, elevated estrogen and progesterone levels are associated with nasal mucosal hyperreactivity. These hormones also induce mucosal swelling, glandular secretion, and dilatation of turbinate capillaries, resulting in worsening of symptoms, especially nasal obstruction.<sup>1, 3, 5</sup>

It is believed that placental growth hormone (PGH) may stimulate mucosal growth and thus induce nasal congestion. Other than that, physiological changes during pregnancy also attribute to symptoms severity. Increased circulating blood volume during pregnancy, up to 40% of pre-pregnancy, is related to increased nasal airway resistance.<sup>5</sup>

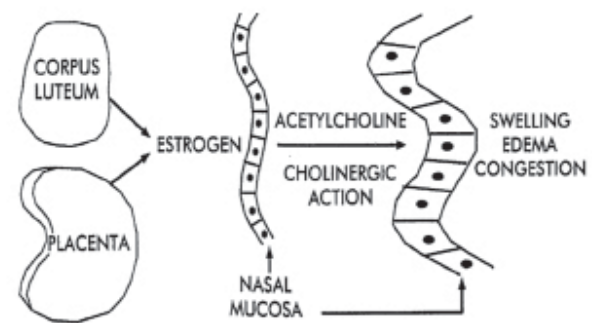
Due to similar symptoms with other inflammatory diseases of the nose, pregnancy rhinitis should be suspected by the exclusion of other causes of rhinitis.<sup>6, 7</sup> Rhinitis symptoms in pregnancy might not be considered as fatal, however, worsen symptoms during pregnancy may impair maternal daily activities and emotional well being. Persistent nasal congestion resulting in sleep disturbance during pregnancy is related to intrauterine growth retardation and lower APGAR scores.<sup>3</sup> Proper management of pregnancy rhinitis must not raise problems, and worth consider the risk-benefit ratio for mothers and infants.<sup>3, 6, 7</sup>

## The Effects of Pregnancy Hormones to Nasal Physiology

Throughout the pregnancy, hormonal changes affect the physiologic nasal cycle in many ways. Pregnancy-related hormonal changes and neuropeptides are causing this alteration to the mucosa of the nose.<sup>8</sup>

### Estrogen

Estrogen production dramatically increases during pregnancy. Estrogen tends to inhibit acetylcholine esterase leading to the production of acetylcholine and induces Parasympathetic activity.



**Figure 1.** Estrogen Effects during Pregnancy<sup>11</sup>

This cholinergic activity causes vasodilatation and oedema to the nasal mucosa.<sup>9, 10</sup> (Figure 1)

In general, estrogen causes nasal turbinates to be edematous, leading to nasal obstruction, nasal discharge flowing into the throat (postnasal drip). Untreated conditions may cause olfactory function disturbance. These rhinitis symptoms also occur in women with birth control pills and hormonal replacement therapy.<sup>12, 13</sup>

Other known effects of estrogen are increased of vascular permeability, protein synthesis, glandular activity and increase the sensitivity of vasomotor properties in the autonomic nervous system. Increase of protein synthesis and hyaluronic acid in the nasal mucosa causes the mucus to thicken and disrupt the mucociliary clearance of the nasal cycle.<sup>9, 10</sup> In addition, increase of blood pooling due to decreased  $\alpha$ -adrenergic smooth muscle tonus in the venous sinusoid, or oedema caused by plasma leakage from the vascular bed to the stroma.<sup>3</sup>

Excessive mucus production gives a sensation to the throat as post nasal drip, thus enhance nasal congestion. Studied nasal mucosa biopsies from pregnant women and those who consumed birth control pills, which he found glandular hyperactivities and increased phagocytosis. Nasal congestion is the initial side effect of high dose estrogen contraceptive pill intake.<sup>14</sup> Conducted a cohort study of 568 patients and found a significant correlation between the incidence of asthma and rhinitis during pregnancy. Furthermore,  $\beta$ -estradiol and progesterone have receptors in nasal mucosa that contribute to the nasal congestion pathophysiology in pregnant women.<sup>12, 13, 15</sup>

The significant rise of the plasma volume in pregnant women occur during the 6 – 8 weeks of gestation and reach its peak at week 32. Volume may increase to 4700 to 5200 ml (45% increase to unpregnant women). This occurrence happened due to an increase of estrogen secretion and renin angiotensin aldosterone system stimulation, which attract water and retain sodium. Water retention produces oedema of the mucosa thus resulting nasal congestion.<sup>11</sup>

Estrogen may trigger an immune reaction through the  $\alpha$ -estrogen receptor on the mast cell. This reaction peaks during the menstrual period, pregnancy, oral contraception consumption and hormone replacement therapy. A hypothesis states that estrogen and progesterone act as antigen bound to a different protein producing Th2 cell, which regulates IgE synthesis and other antibodies. The antibody linked to mast cells as well as appropriate antigens (hormone or metabolites) will cause degranulation of the mast cell or basophil. Thus releasing histamine, Th2 cytokines, and leukotrienes.<sup>16, 17</sup>

### Progesterone

There was no difference in blood progesterone level between women with and without pregnancy rhinitis. Other study showed an increase of circulatory blood volume, possibly from vasodilatation occurring due to the increased level of progesterone in pregnant women, which may induce nasal congestion.<sup>2</sup> Nasal vascular pooling from smooth muscle relaxation related to the increase of progesterone. An increase of vasoactive intestinal peptide (VIP) release,

stimulated by progesterone and oxytocin, may enhance nasal congestion. Progesterone-related fibroblast in the nasal mucosa may also affect the extracellular matrix.<sup>10</sup>

### Prolactin

Production of prolactin by pituitary increases during pregnancy, which suggests the possibility of its role in the pathogenesis of pregnancy rhinitis; however, this is contradicted by the absence of sinus pathology in patients who have prolactinomas. Furthermore, bromocriptine and quinagolide reduce prolactin production which eventually develops nasal congestion.<sup>3</sup>

### Neuropeptides

A vasoactive intestinal polypeptide (VIP) is associated with other forms of rhinitis, and a possible mediator for nasal mucosa vasodilation, which is responsible for nasal congestion during pregnancy. Nasal biopsies of postmenopausal women showed an increased immunopositivity for estradiol, estradiol receptor, VIP, and substance P (SP) after six months of hormone replacement therapy. Whereas, there was a reduction in neuropeptide Y (NPY). Nasal application of hormone replacement therapy induced stronger VIP changes than did the transdermal application. Mucociliary transport time and subjective nasal congestion decreased, but anterior rhinomanometry was unchanged. The investigators proposed that estrogen action in the nasal mucosa is mediated by neuropeptides an increase of gland secretion and vasodilatation by VIP and SP and a decrease of NPY-induced vasoconstriction.<sup>3</sup> VIP relaxes the blood vessels to the upper airways, trachea, bronchi, and pulmonary vessels.

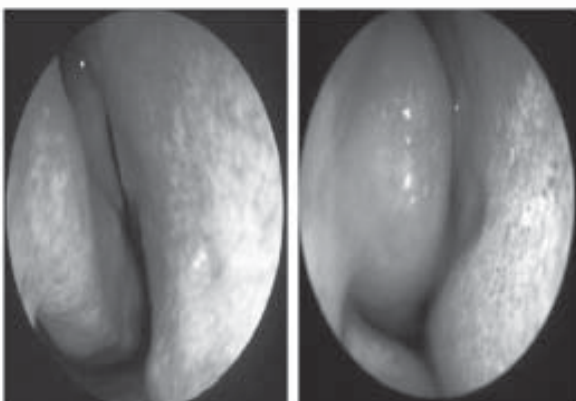
Innervation of the nasal mucosa is mainly organized and complex. The autonomic system regulates the mucosal vasculature and glandular secretion. The efferent nasal reflex arc consists of sympathetic and parasympathetic nerves. Parasympathetic nerve stimulates the release of acetylcholine, norepinephrine, and VIP. Postganglionic parasympathetic nerve innervates serous and mucous glands, arteries, veins and arteriovenous anastomoses. The distribution of VIP-immunoreactive fibres corresponds to the cholinergic distribution system. VIP stimulates

serous cell secretion, dilates nasal vessels, and may also regulate mucociliary clearance in the nose.<sup>18</sup>

The mechanism of hyperactivity of the nose remains unknown. The proposed hypothesis is increased permeability and increased the sensitivity of sensory nerve endings and imbalance of autonomic nerve regulation caused by changes of the nasal mucosa neuroreceptors.<sup>18</sup> Substance P is produced by the afferent sensory neurons of the trigeminal nerve within the nasal mucosa. Neurotransmitter promotes vasodilatation, increase blood vessels permeability, and hypersecretion of submucosal glands, leading to all sorts of nasal symptoms. The exact role of this neuropeptide in pregnancy rhinitis remains unclear.<sup>19</sup>

Small-diameter of the unmyelinated sensory fibres which are extensively branched, densely innervate the walls of submucosal vessels and glandular acini to form the neurosecretory varicosities within the vascular and glandular area. Neuronal wave evoked by histamine immediately extends to the peripheral sensory neurons, and the central, brain, as well. Hence, various neuropeptides are released from the nerve endings into the spaces near submucosal vessels and gland to elicit its rapid reactions.<sup>19</sup>

### Placental Growth Hormone



**Figure 2.** nasoendoscopic finding right inferior turbinate in pregnancy rhinitis pre (right) and post (left) decongestant.<sup>3</sup>

Human Growth Hormone (hGH) is secreted in an episodic burst in low levels between peaks. This pattern is later replaced by continuous secretion of Placental Growth Hormone (PGH) after the first trimester of gestation. That placental

growth hormone (PGH) was significantly high during pregnancy. A significantly higher level of PGH in women with pregnancy rhinitis group on all occasions throughout the pregnancy. Presumably, PGH stimulates nasal mucosa similar to progesterone, thus inducing pregnancy rhinitis. PGH has a similar effect as progesterone in pregnancy, which is peripheral vasodilatation and increases extracellular volume.<sup>20</sup> The mechanism is still unknown; further study is warranted.<sup>3, 20</sup>

### Risk Factors

According to a questionnaire study done, the incidence of pregnancy rhinitis is significantly higher among smoking women than non-smoking women (odds ratio: 1,7; CI 95% 1,1-1,5)<sup>2,3</sup>. Thus, smoking is considered an irritant agent that most probably stimulates nasal congestion. In vitro test to 10 airborne allergens in 165 pregnant women, in which 83 women had pregnancy rhinitis<sup>14</sup>. Overall sensitization was not increased in women with pregnancy rhinitis, yet sensitization to house dust mites frequently occurred in this group. Therefore, subjects with a high level of IgE to house dust mites are considered prone to develop pregnancy rhinitis. However, to differentiate pregnancy rhinitis to allergic rhinitis in pregnancy is still a challenge.<sup>2, 3, 14</sup>

### DIAGNOSIS

The diagnosis of pregnancy rhinitis is made by history taking, consisting thorough information regarding symptoms and physical examination to eliminate other cause of other nasal disorders. This shall exclude allergic rhinitis, vasomotor rhinitis, septal deviation, polyposis, rhinosinusitis and many more. It is difficult to differentiate allergic and nonallergic rhinitis. The common triggering factor of nonallergic rhinitis may be weather or temperature changes, food, perfume, strong odour and smoke. Additional symptoms may include allergic conjunctivitis (itching, watery, redness and swelling of the eye).<sup>21</sup>

A comprehensive head and neck examination starting with a simple rhinoscopy or nasoendoscopic examination to exclude other cause of rhinitis (Figure 2). The mucosa of the nasal and nasal turbinates may appear swollen and covered with serous to seromucoid discharge. There are no other specific further findings (laboratory or other means) to diagnose



pregnancy rhinitis. Pregnancy rhinitis is diagnosed based on subjective findings of symptoms and physical examination.<sup>21</sup>

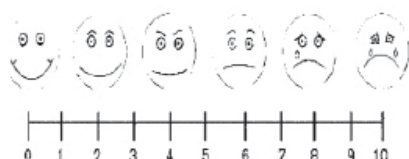


Figure 3. Visual Analog Scale (VAS)<sup>22</sup>

The evaluation of subjective nasal obstruction using a Visual Analog Score (VAS) and Nasal Obstructive Symptom Evaluation (NOSE) scale may assess the quality of daily life (Figure 3 and Table 1). Both tools are highly specific and sensitive, validated by previous studies on many other nasal pathologies. VAS and NOSE scale are patient-centred quantitative diagnostic tools. VAS score more than 5 and NOSE scale more than equal to 5 in pregnant women presumably to be pregnancy rhinitis.<sup>22</sup>

Table 1. Nasal Obstructive Symptom Evaluation Scale<sup>23</sup>

	Mild	Moderate	Fairly bad	Severe
Nose obstruction and stuffiness	0 1	2	3	4
Nose obstruction	0 1	2	3	4
Trouble breathing through my nose	0 1	2	3	4
Trouble of sleeping	0 1	2	3	4
Unable to get enough air through my nose during exercise or exertion	0 1	2	3	4

Over the past month, how much of a problem were the following conditions for you? Please circle the most correct response.

Discharge Inflammation Polyps/Oedema (DIP) score is a clinician-based examination validated by previous researches (Table 2). DIP score quantifies nasoendoscopic findings (more than 5 to be considered as pregnancy rhinitis). A correlation between VAS, NOSE, and DIP scores toward gestation in evaluating pregnancy rhinitis.<sup>4</sup>

Table 2. Discharge Inflammation Polyps/Edema (DIP) Scoring System<sup>24</sup>

	Absent 0	Moderate 1	Severe 2
Discharge			
Inflammation			
Polyps/Edema			

Nasal congestion may also be objectively assessed by rhinomanometry, acoustic rhinometry, or peak nasal inspiratory flowmetry (PNIF). Those examinations assess nasal congestion by measuring nasal resistance, nasal volume, and nasal airflow.<sup>24</sup>

## DIFFERENTIAL DIAGNOSIS

Pregnancy rhinitis is a subtype of non-allergic non-infectious (NANIR). Allergic rhinitis shows similar symptoms to pregnancy rhinitis, but the underlying pathology occurred due to immunoglobulin E-mediated hypersensitivity. A definitive diagnosis of allergic rhinitis is established by skin prick test or specific IgE serology results.<sup>2, 3, 25</sup>

Rhinitis medicamentosa is another differential diagnosis but may also become a complication of pregnancy rhinitis. Most women do not directly disclose a history of prolonged usage of intranasal decongestant. Therefore it is essential to obtain this information in the history taking. Healthy individuals with rhinitis medicamentosa no longer have nasal congestion in 2 days after they stop using decongestants. During pregnancy, if congestion persists for more than a week or so after reducing intranasal decongestant, the diagnosis should be pregnancy rhinitis.<sup>3</sup>

Other differential diagnosis includes upper respiratory tract infection and anatomical variation (septal deviation and hypertrophic turbinates) which disrupt the mucociliary clearance. Infection may occur due to a virus which causes direct damage to the nasal epithelial barrier and indirectly by hyperactivity of nasal mucosa, thus disrupts mucociliary clearance. It is important to exclude sinusitis by the clinical finding of purulent discharge in the middle meatus, facial pain, and olfactory dysfunction. Sinusitis during pregnancy (common in the second trimester) may not improve with conservative treatment such as nasal saline irrigation. Secondary bacterial infection may also occur. Typical organisms found in such conditions are *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*.<sup>26</sup>

## IMPACT OF PREGNANCY RHINITIS

Impact of pregnancy rhinitis to the fetus is indirectly related to sleeping disturbance

experienced by pregnant women. Besides nasal congestion due to placental hormones, lying down in bedtime (supine position) also cause more nasal congestion due to the effect of gravitation. Difficulty in breathing through the nose increases the risk of snoring and mouth breathing during sleep. Twenty three percent of 502 postpartum women reported having to snore at the last weeks of their pregnancies. Snoring gives a higher risk of developing hypertension, pre-eclampsia, intrauterine growth retardation (IUGR), and low APGAR scores. Mouth breathing in pregnancy rhinitis affects lung vascular tonus or mother/ and fetus oxygenation. Pregnancy rhinitis might induce the presence of obstructive sleep apnea (OSA).<sup>2,3</sup> Physiologically, inhaled nitric oxide, which is highly produced inside the maxillary sinuses, decreases lung vascular resistance and increases lung oxygenation. Nasal congestion might disturb nitric oxide production.

Another study stated that pregnancy rhinitis impaired women's quality of life and might cause OSA, which in turn disturb pregnancy outcome<sup>25</sup>. Quality of life impairment was most significant at the third trimester than the second trimester. In addition, there is a significant difference in the quality of life between women with and without pregnancy rhinitis. Prolonged nasal congestion may lead to sinusitis development. Furthermore, it may cause cognitive and concentration impairment, fatigue, and headache. Pregnancy rhinitis is also associated with increased anxiety. Mouth breathing, on the other hand, causes dry mouth and decreases saliva secretion, leading to dental caries due to loss of salivary protection effect.

Quality of life in pregnancy rhinitis has not been fully evaluated, yet in severe cases, women showed difficulty in achieving optimum respiratory needs during the delivery process. Nasal congestion acts as a potential risk in hampering fetal growth and development by gradual hypoxia.<sup>27</sup>

## MANAGEMENT

### Conservative treatment

In the conservative approach, it is essential to elaborate treatment options and goals to women with pregnancy rhinitis, which is to alleviate symptoms rather than cure the disease.

Doctors must ensure pregnant women that nasal congestion is a common occurrence during pregnancy. Such information should be given at the first antenatal care visit, along with several treatment options to choose.<sup>2,3</sup>

### Physical Exercise

Physical exercise has been known to have a decongestant effect on the nasal mucosa. Head elevation at 300–450 when laying down reduce the likelihood of vena cava syndrome and snoring. Another mechanical mean is using dilator fonostrils. This device dilates the narrowest part of the upper airways that is valve area of the nose. External type dilator may improve nocturnal nasal congestion related to breathing in pregnant women, while internal type dilator may reduce snoring in men as effective as a nasal decongestant. The adverse effect from dilator usage would be local irritation of the skin due to pressure.<sup>28</sup>

### Nasal Saline Irrigation

Nasal saline irrigation is effective in improving symptoms, by repairing mucociliary clearance, reducing mucosal oedema, reducing inflammatory mediators, and cleaning mucus or triggering agents. Nasal irrigation is performed using isotonic saline solution 30–500 ml (average 200–250 ml), pH range is 6.2–8.4. Higher volume irrigates a wider area in the nasal cavity. Hypertonic solution (3% sodium chloride solution) is effective for irrigating thick mucus. Depending on device and volume, nasal irrigation on average is performed in 1 minute with compression pressure  $\geq 120$  mbar to reach areas of the nasal cavity.<sup>29</sup>

## Pharmacology Treatment

A pharmacological agent is an option when conservative therapy fails to reduce symptoms of pregnancy rhinitis.

### Decongestant

Decongestant is a vasoconstrictor agent effective to reduce nasal obstruction. Most systemic decongestant (phenylephedrine, pseudoephedrine, and phenylpropanolamine) is classified as category C by the United State Food and Drug Administration (US FDA). One study

found 206 cases of gastroschisis due to systemic decongestant during pregnancy, although there has been no study in the effectiveness of systemic decongestant for pregnancy rhinitis.<sup>3</sup> In unpregnant women, a decongestant may cause systemic side effects such as high blood pressure, palpitation, decrease appetite, tremor and sleep disturbance.<sup>9</sup>

Topical decongestants (oxymetazoline and phenylephrine) work rapidly to relieve nasal congestion. Pregnant women tend to overuse nasal decongestant in the long term because pregnancy rhinitis is a continuous condition. This will lead to unresolved rhinitis medicamentosa postdelivery. A topical decongestant will occupy  $\alpha$ -adrenergic receptors more. Hence the autoregulation system will cause rebound mucosal oedema and worsen congestion.<sup>10, 11</sup> Benzalkonium chloride, a common preservative in topical decongestant also aggravate nasal congestion. Usage of topical decongestant once daily at night for more than four weeks may develop into rhinitis medicamentosa.<sup>27</sup> Therefore the recommended dose is a short term use of maximum 5-10 days at a lower dosage, unilateral and alternating nostril administration in the evening.<sup>3, 28</sup>

### Glucocorticoids

The intranasal steroid gives a very responsive therapeutic effect on managing all forms of rhinitis (allergic rhinitis, medicamentosa, NAR and sinusitis). Intranasal steroid lowers the need for a systemic steroid, and it has an insignificant effect on pregnancy rhinitis. A RCT study by Ellegard et al<sup>2</sup> showed insignificant effects of fluticasone propionate nasal spray given eight weeks to pregnancy rhinitis, observed from symptoms or acoustic rhinometry data. That study also found no impact on cortisol level or fetal growth shown in the ultrasound. All currently available intranasal steroids have extremely low bioavailability, hence hardly give systemic side effects. Regardless, US FDA classifies mometasone furoate, fluticasone furoate, fluticasone propionate, and triamcinolone acetonide as category C. According to the Swedish Medical Birth Registry, there is no increment of congenital malformation incidence in rhinitis patients using budesonide inhalation in the early gestation. Hence it is classified as category B.<sup>2, 3, 28</sup>

Long term and repeated systemic corticosteroid usage should be avoided due to its adrenal suppression and other systemic side effects. A short-term of systemic steroid, less than two weeks, may give temporary relief. High dose corticosteroid intake gives many risks in the first trimester, for example, fetal blindness, lung oedema, uterine contraction inhibition, and fluid overload.<sup>28</sup> Thus, systemic corticosteroids generally should be avoided during pregnancy, except in serious threatening condition with benefit/risk ratio concerns.

### Antibiotic

Antibiotic is not indicated in pregnancy rhinitis, although that would not be the case when bacterial sinusitis occurred. Intensive high dose of antibiotic is warranted, Beta-lactam dose should be increased by 50% because of renal clearance increase during pregnancy and to reach minimum inhibition concentration. Category B antibiotics, other than Beta-lactams, include penicillin, cephalosporin, aztreonam (monobactam). Imipenem is classified as category C.<sup>3</sup>

### Antihistamine

Antihistamine is indicated if histamine-related symptoms are suspected, particularly effective for relieving sneezing and nasal itching.<sup>3,5</sup> Antihistamine has been used for a long time and known to have no adverse effect on the fetus. However, classic or 1<sup>st</sup> generation antihistamines (i.e. chlorpheniramine, triprolidine, diphenhydramine, cyproheptadine, promethazine, ketotifen) show to have side effects due to their action. Such side effects are drowsiness, dry mouth, and increase appetite.<sup>12</sup> Classic antihistamines are associated with oral clefts. Second and new generation antihistamines (i.e. loratadine, cetirizine, fexofenadine, desloratadine, levocetirizine) are more preferred due to lack of such side effects and no cardiac effects. This group of antihistamine is also clinically proven to have anti-inflammatory effects, aside from a known H1-histamine receptor blocker. Cetirizine and Loratadine are classified as category B.

### Other Methods of Management

Nasal Continuous Positive Airway Pressure (CPAP) is indicated in pregnancy rhinitis with severe

obstructive sleep apnea (OSA). In unpregnant patient, nasal CPAP is highly effective as shown by the improvement in polysomnography results. Pressure adjustment must be made in pregnant women. A study shows that nasal CPAP significantly reduces the nocturnal blood pressure in pre-eclamptic women without OSA.<sup>3</sup>

## Surgery

Surgery will only be in consideration in cases with worst prognosis due to failure of conservative, pharmacological therapy, and other noninvasive measures, such as CPAP. It should be thoroughly considered that pregnancy rhinitis is a self-limiting disease in a certain period.<sup>3</sup> For refractory nasal obstruction due to hypertrophic turbinate, less invasive surgical measures include electrocautery, cryotherapy, laser or radiofrequency may be applied to reduce inferior turbinate volume.

## CONCLUSION

Researchers describe pregnancy rhinitis as nasal congestion that occurred in 6 weeks or more of the last final trimester of pregnancy, without a sign of respiratory infection and known allergy, which may resolve completely in 2–4 weeks postpartum. The most bothersome symptom is nasal obstruction, and other symptom includes watery rhinorrhea. Its pathomechanism remains unclear, although it is believed that estrogen, human chorionic gonadotropin hormone (HCG), human placental lactogen (HPL), and placental growth hormone (PGH) play roles. Risk factors include smoking and sensitization.

There is no drug of choice to manage pregnancy rhinitis. Women with pregnancy rhinitis should receive proper education regarding their conditions. Although most pregnancy rhinitis spontaneously resolved after giving birth, symptoms may significantly decrease quality of life, and medical attention is needed. Management includes conservative and pharmacology treatment, which aimed to relieve nasal congestion and other symptoms. Nonetheless, pregnancy is a specific condition that needs specific consideration. The final goal is to have an optimal outcome for both mother and baby.

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